

ACUTE SUPPURATIVE PERICARDITIS* WITH AN INITIAL LEUKEMOID BLOOD PICTURE

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IT is of interest to note that Galen first observed pericarditis in animals in 131-201 A. D. It was not until 1649, however, that Riolan first advocated pericardiotomy by trephining the sternum. The first successful pericardiotomy was performed by Laennec in 1819. Then, in 1844, Hilsman was the first to perform pericardiotomy for suppurative pericarditis. Many others discussed the various aspects of pericarditis up to that time, and since then still others have worked out the finer points in pathology, diagnosis, and treatment.

The case we are about to report was trying and puzzling from the beginning. The important symptoms, on the patient's entrance to Monterey County Hospital at Salinas, were fever, pain, and swelling in the right neck. For one so sick, as you will soon see, together with the fact that this swelling subsided without treatment in thirty-six hours, we were misled at first completely. The blood count showed a mild secondary anemia, with a low leukocytosis, *i. e.*, 10,500, with 5 per cent myeloblasts and myelocytes. There were a great many nucleated red cells and many polychromatophylic red cells. This type of white count continued for one month. Again we were puzzled to know whether this was an aleukemic phase of myelogenous leukemia. As Krumbhaar states, "The recognition of many acute cases which have only a mild leukocytosis, or even a leukopenia, coupled with fever and some localized signs of infection, may make immediate diagnosis a difficult or impossible feat." One may refer here to the number of conditions that give a leukemoid blood picture, *i. e.*, (1) measles and pertussis; (2) acute infectious lymphocytosis; (3) acute infections with hemorrhage; (4) terminal septicemia with myeloid leukemia; (5) bone-marrow intoxication, with terminal blood picture; (6) agranulocytosis, like acute leukemia; and (7) myeloma, resembling acute leukemia. Two cases were given with terminal leukemic pictures, and at autopsy they could not be distinguished from acute leukemia and acute gastrocolitis and Banti's disease, respectively.

REPORT OF CASE

This patient, a young American male, was admitted, walking to the Monterey County Hospital, on the morning of May 20, 1937. He was twenty-four years of age, weighed 170 pounds and was about twenty pounds under his normal weight. His chief complaint was a painful swelling on the right side of the neck, and he stated he had had a sore throat for about twenty-four hours before coming to the hospital.

Family History.—Native American stock from Oklahoma, otherwise not remarkable.

Past History.—The usual childhood diseases, but other ailments were not remembered. These are irrelevant to his present illness.

Present Illness.—He became acutely ill about a month prior to his admission to the hospital, suffering from severe pains in his throat and chest, and a shortness of breath, with moderately productive cough. He states that he remained in bed for about ten days, after which he would get up and walk about his room, but had to return shortly to bed as he tired easily. His throat became sore a few days before coming to the hospital, and the swelling came on in his neck one day before.

Physical Examination.—Temperature, 100; pulse, 100; respiration, 20. Patient appeared acutely ill. *Skin:* moist and slightly cyanotic. *Eyes:* pupils equal and active to light and accommodations. *Eye grounds:* normal. *Mouth:* oral hygiene poor, with moderate gingivitis and pyorrhea, along with many cavities in the teeth. *Throat:* mucous membrane injected, edematous, and slightly cyanotic. *Tonsils:* large and cryptic. *Neck:* soft and somewhat rubbery, slightly edematous, and tender swelling over the right anterior and posterior triangles. Few small, palpable anterior cervical lymph nodes. *Thyroid:* normal. *Heart:* A. C. D. widened to the anterior axillary line on the left. P. M. I., faint in the fifth left interspace, 17 centimeters from M. S. L. The right border is 4 centimeters from M. S. L. The aortic dullness is 8 centimeters. *Heart sounds:* distant, no murmurs or thrills; rate regular, 100 to 126. A 2 greater than P 2. Blood pressure: 100/60. *Lungs:* the voice sounds and breath sounds are increased over the anterior upper chest. A pleural rub is present on the right above the third rib. *Breath sounds and voice sounds* are bronchial over both upper posteriorly. Tactile fremitus is decreased in the posterior mid-right, and flat at the right base. The percussion note is also flat on the left posterior below the spine of the scapula. *Abdomen:* soft, spleen not palpable, but liver is 6 centimeters below the costal margin, and to the fourth rib above. No masses, fluid or tenderness made out. *Genitals:* normal. *Rectal:* negative. *Prostate:* normal. *Extremities:* muscularity good, no clubbing of the fingers, but slight cyanosis on the tips of the fingers and toes. *Neurological:* cranial nerves all normal. *Reflexes:* upper and lower extremities normal. Romberg, Kernig, and Brudzinski, all negative.

Laboratory Reports.—*Urinalysis* showed a normal specific gravity with a trace of albumen, and an occasional white blood cell and red blood cell with a rare granular cast. This continued throughout his hospitalization.

Blood.—The first count showed: Hemoglobin, 88 per cent; red blood cells, 4,140,000; white blood cells, 10,600; polymorphonuclears, 68 per cent; lymphocytes, 27 per cent; eosinophils, 1 per cent; myeloblasts and myelocytes, 4 per cent; smears showed many basophilic red blood cells, no stippling, many nucleated red blood cells. The myeloblasts and myelocytes disappeared by June 30, 1937. A secondary anemia developed by August 11, 1937, *i. e.*, hemoglobin, 44 per cent; red blood cells, 2,450,000. The white blood cells fluctuated between 11,000 and 13,000. With the above anemia it reached 8,200 with an essentially equal differential. The patient's blood count following therapy gradually rose to normal by November 19, 1937.

E. K. G.—*Auricular rate*, 130; *ventricular*, 130; *P. R.* 0.12; *Q. R. S.* 0.06; normal axis deviation. *Sinus*, regular rhythm. Inverted T, one and two. T 3, diaphasic; T 4, upright and slightly high take-off. Complexes in almost iso-electric. All other complexes low.

Sputum.—May 24, 1937. Tubercle bacilli, none. Many Gram-positive cocci in pairs and masses.

Bacteriology.—Blood cultures were always negative. The pericardial fluid, on May 25, 1937, was serosanguineous and contained many pus cells. This showed, on culture, hemolytic *Staphylococcus aureus*. One week later the findings were the same. On June 5, 1937, showed a clear sterile fluid. On June 23, 1937, left pleural fluid was cloudy but sterile. This became purulent with many pus cells on August 5, 1937, and a culture of hemolytic *Staphylococcus aureus*. With treatment this became sterile six weeks later.

X-ray showed, on May 23, 1937, a massive pericardial effusion and a resolving pneumonia of the right lower

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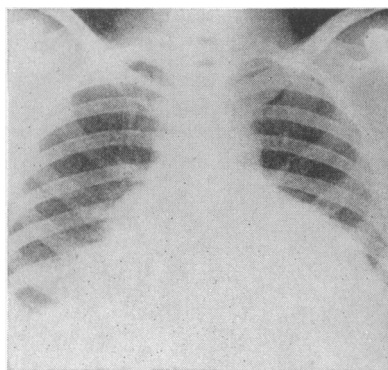


Fig. 1

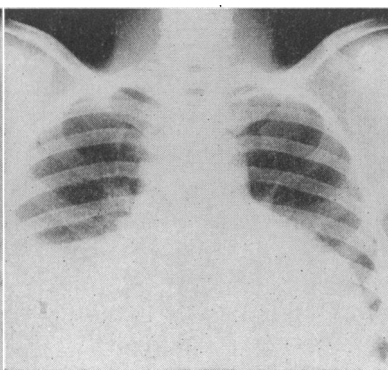


Fig. 2

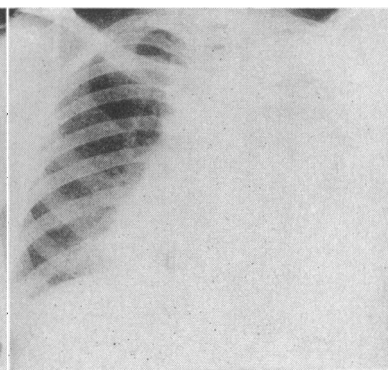


Fig. 3

lobe. Ten days later the pericardial effusion had increased and a large left pleural effusion and a small right one developed. First postoperative films, on July 29, 1937, showed massive left pleural effusion. Heart shadow not visualized well. On August 5, 1937, heart shadow was normal in size with hydropneumothorax left. Right chest entirely clear. Six weeks later, following second left rib resection, showed no fluid, but thickened pleura. One week later, September 22, 1937, showed left lung 80 per cent expanded. Heart shadow and right chest normal. Serial films from this date, November 26, 1937, showed gradual reexpansion of left lung and marked thickened pleura on the left. Normal heart size.

PATHOLOGY

The essential pathology of pericarditis has been shown to be an inflammatory reaction to both the visceral and parietal pericardium, with thickening and often an increase in size of the parietal layer. Delafield and Prudden state "that in this form of exudative pericarditis there are usually more or less serum and fibrin mingled with pus cells, and often red blood cells. The process may start as a serofibrinous inflammation. It is apt to occur as an extension of an infectious process in the neighborhood, or as part of a general pyemic process." The organism may be in the pericardial wall (seen on microsection), but not in the purulent fluid. The heart musculature may or may not show an inflammatory attack. The offending organism, so states Branch,³ may be according to percentages in cases, *i. e.*, pneumococcus 35 per cent, in acute cases; organisms of rheumatic fever 30 per cent; staphylococcus, streptococcus, and tuberculosis, about 15 per cent. Hemolytic staphylococcus pericarditis has the highest mortality. Noninfectious terminal cases, 20 per cent, such as uremia, diabetes, anemia, leukemia, and coronary heart disease. As for the source of the pericarditis with or without suppuration,^{5,6,7} intrathoracic infectious disease was apparently the primary condition in 74 per cent of cases. Infections were the etiologic factor in 98.2 per cent, with a predominance of males.*

Various conditions may be considered in the etiology, such as malignancy, chronic passive congestion, cardiac infarction, hypertension, bronchopneumonia, rheumatism, myxedema, subacute bacterial endocarditis, tuberculosis, otitis media, mediastinitis and laryngeal edema, myelogenous, leukemia, nephritis, liver abscess, infectious arthritis, goiter, lues, influenza, measles, tonsillitis, pleurisy, puerperal sepsis, whooping cough, osteo-

myelitis, esophageal perforation, pyemia, gunshot and stab wounds, gangrene of the feet; the pericardial fluid may be serous, serofibrinous, turbid, serosanguineous, bloody and purulent, or it may be encapsulated.

TREATMENT

When the correct diagnosis of acute suppurative pericarditis was made clinically, the next step was to determine the correct method of treatment. The anatomic, physiologic, and pathologic bases for treatment of pericarditis have been successfully covered in various texts and articles on the subject. It is to be remembered that a collection of purulent material, due to infection in any closed sac, must be considered as an abscess and, as such, evacuated. This may be accomplished by two methods, namely, paracentesis pericardii, *i. e.*, "closed drainage," which may, and generally will, of necessity be repeated, with or without irrigation of the sac, with various antiseptic agents; or pericardiotomy, *i. e.*, "open drainage" with continued drainage with or without the use of various antiseptic agents for irrigation. The advantages and disadvantages of both methods have been discussed by various authors, and the interested reader is referred to the bibliography attached for details of the various methods.

Briefly, the mortality of purulent pericarditis by expectant methods, or by aspiration, is above 70 per cent, and most authors would place this figure at 100 per cent. Shipley and Winslow, in 1927, collected 128 cases treated by pericardiotomy, with recovery in over 55 per cent. In 1935, they collected ninety-nine cases more, with recovery in over 49 per cent. These two articles in particular have given an excellent review of the literature, as have writings by other authors, and it is unnecessary to repeat the many excellent statistics here. It would seem, however, that any method which would reduce the mortality from 100 to 50 per cent at least offers a great deal, particularly when one considers that some 80 to 85 per cent of all cases of purulent pericarditis are in young adults under thirty, and 70 per cent of all cases are in males. Aspiration, although a simple procedure, is not without its dangers, and when one considers the difference in mortality results with open pericardiotomy, the evidence must be in favor of the latter procedure. Doctors Shipley and Winslow have said: "If unrecognized, purulent pericarditis kills

* Slide 1. Marked pericardial effusion.

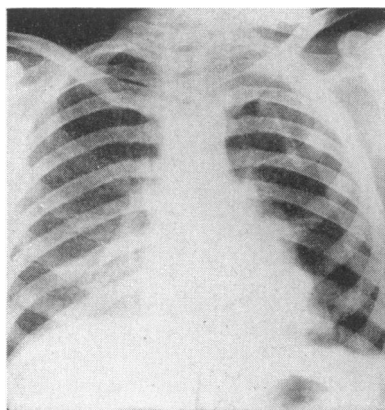


Fig. 4

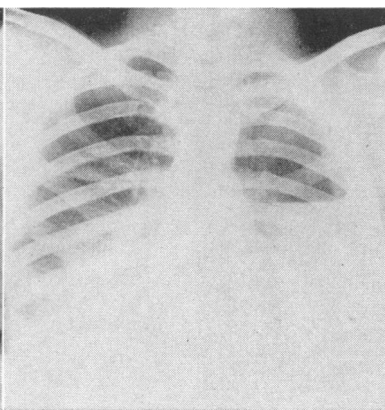


Fig. 5

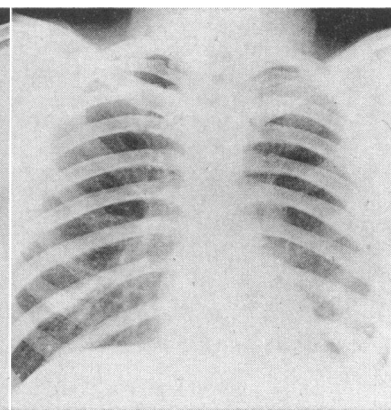


Fig. 6

the patient" and "the patient, in whom the condition is diagnosed and who is treated expectantly, or by therapeutic aspiration, fares no better." Operative attack is the only method at our disposal which offers any chance to cope successfully with the condition. To repeat, as in abscess elsewhere, adequate drainage would seem to be the crux of the treatment of purulent pericarditis, and it is of paramount importance in the prevention of hertz-tamponade with stoppage of the heart and death. It is not our purpose to discuss here the various means of approach to the pericardial sac, each of which will vary according to the personal preference of the surgeon, but among these may be mentioned the right parasternal approach, with resection of the fourth, fifth, or sixth costal cartilages; the left parasternal with resection of the fifth, sixth, or seventh costal cartilages; the transternal approach, or the chondro xyphoid approach. Of these, the left parasternal approach, with resection of the sixth or seventh costal cartilages, seem to have the most advocates. Other factors to be considered are those of adhesions, which may cause pocketing of the pus and which will require breaking up to secure proper drainage of the dependent pockets, or recesses of the pericardial sac. The importance of the prevention of soiling and contamination of the pleura cannot be stressed too strongly, as this adds another hazard to the prognosis in an already dangerously ill patient. The mortality of pericarditis complicated by, or subsequent to empyema, is much higher. Organisms may cross the barriers from the pleura to the pericardium or vice versa, without being due to surgical contamination, and indeed would be difficult to avoid, but the seeding of the pleura by the surgeon certainly should not occur.*

In our case the patient had a clear and sterile pleural effusion bilaterally, more on the left than on the right. The right effusion was cleared up and remained so by a single aspiration. It might have been wiser to perform open drainage a little earlier in the disease; but, as previously noted, there were several complicating factors, making the absolute diagnosis somewhat uncertain until just before operation. Obviously at this time the patient was not doing well, and we felt to temporize

longer would reduce the chances for recovery, although statistics show little difference between mortality in early or late operations. The operation was attended with little or no shock, and although the patient had a somewhat rapid and irregular pulse, perhaps due to slight irritation from the soft rubber drain, his convalescence was uneventful, with good drainage and rapid healing of the wound.* Although the pleural cavity was not opened, the sterile pleural effusion subsequently became contaminated with the same organism six weeks later, and eventually required open drainage with rib resection,† as repeated aspiration failed.

Although adequate drainage was established by the first rib resection, the wound closed too early, and a second operation was required, with subsequent irrigation with azochloramid solution. About eight weeks passed between the pericardiotomy and the first rib resection, and five weeks between the first and second rib resection. Unfortunately the sections of the rib were disposed of before smears could be made of the bone marrow to check the early blood findings. The empyema wound was completely healed sixty-nine days after the second operation, at which time the patient had been up and around for about four weeks.‡ At the time of discharge from the hospital the patient was perfectly well and exhibited no evidence of cardiac disability, *i. e.*, no dyspnea, no edema or subjective symptoms of pericardial or thoracic pain or distress. There was no evidence of constricting pericarditis or adhesion, such as edema, enlarged liver, venous distention, cyanosis, or pulmonary congestion.

He was last seen on July 14, 1938, eight months after his discharge from the hospital, at which time he reported he was working without any difficulty and with only occasional slight dyspnea after extra exertion and occasional aching over the pericardium. His physical examination was essentially negative, with a slight elevation of the blood pressure, 145/100. Fluoroscopic examination revealed a small pericardial adhesion posteriorly, and a little pleural thickening on the left. He is obviously

* Slide 3. Right effusion cleared; left pleural effusion formed.

† Slide 4. Pericardial and left pleural effusions drained surgically.

‡ Slide 5. Left empyema reformed due to poor drainage.

* Slide 2. Pericardial and right pleura effusions.

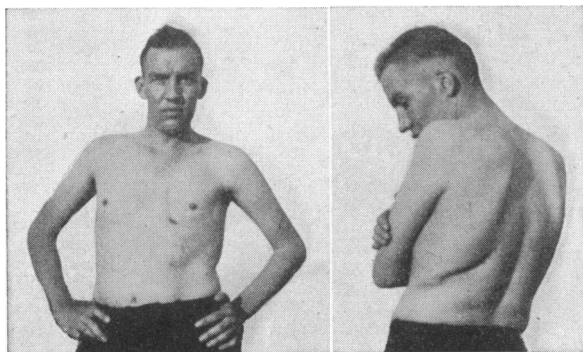


Fig. 7

Fig. 8

well and active now. It is too early to state if he will require cardiolysis. At the present time we feel that in all probability he has a fair chance of continuing well without further procedure.*

OPERATIVE PROCEDURES

Operation: By Dr. W. L. Rogers of San Francisco and Dr. John C. Sharp of Salinas, California, on June 10, 1937.

Local anesthesia with three-fourths per cent novocain and adrenalin. A two-inch portion of the left sixth rib was resected anteriorly, along with a portion of the sternocostal cartilage. Incision into the pericardial sac without entering the pleural cavity. A soft rubberdam drain was inserted into the wound with drainage of much bloody pus.

In a communication, Dr. W. L. Rogers stated that he had seen the patient for the first time on the morning of June 10, 1937. "The purulent nature of the pericardial effusion had already been investigated and found to contain staphylococci. The left-sided pleural effusion had not been tapped. Upon doing this by means of a posterior approach and clear amber fluid being obtained, an operative approach was easily determined. After the removal of a portion of the sixth rib and its cartilage, the bulging parietal pleura was reflected backward and this portion of the wound packed with iodoform gauze. This was left in place following the operation. A rather large opening was then made into the lower anterior border of the pericardial sac, after pus had been obtained with an aspirating needle. A portion of a surgical rubber glove was used as a drain. This proved very efficacious, as it was soft—being unlikely to irritate the heart, and yet affording a constant portal for drainage. The wound was left open, the soft tissues being loosely packed with iodoform gauze. After eight days the drain was removed. This proved to be sufficiently long and quite in contrast to our experience with pleural exudates.

"The most recent films of the patient reveal a relatively normal heart and pericardial outline. I believe we may safely say he will very likely be spared any late complications due to changes in the pericardium. It would seem to me this factor is

due to the acute pyogenic nature of the disease, in this case, as in contrast to others, having a slowly progressive course with the tubercle bacillus, or some other specific organism as the etiologic factor.

"As these cases are comparatively very rare in our smaller clinics, I wish to congratulate the authors in their successful management of this very interesting but very sick patient."

COURSE

For the first ten days the patient passed an essentially normal convalescent period. At this time the pericardial drain was removed. However, drainage continued freely from the pericardial sac. Temperature and pulse rate remained somewhat elevated. Repeated aspirations of the left pleural cavity yielded large amounts of clear, straw-colored, sterile fluid up to July 28, 1937. On August 4, 1937, temperature had risen to 102 degrees Fahrenheit, and thoracentesis of the left pleural cavity revealed an empyema with the identical organism of hemolytic *Staphylococcus aureus*. On August 10, 1937, with local anesthesia, two inches of the left tenth rib in the posterior axillary line, were resected, and a soft rubber tube was inserted and the wound left wide open. In the meantime the liver and spleen, which had been enlarged, gradually returned to normal. The tube was removed in about two weeks and the drainage continued well. The sinus gradually closed. On September 18, 1937, a second rib resection was performed, because the drainage had practically stopped and the pleural cavity had again filled with pus. At this time a portion of the eleventh rib was resected. Following this the empyema cavity was irrigated with normal saline and 1:1000 azochloramid. The tube was removed in about three weeks and the empyema cavity gradually decreased in size with full expansion of the lung and closure of the sinus by November 16, 1937. On November 23, 1937, the general condition of the patient was excellent: he gained in weight above his normal 194½ pounds; blood pressure 130/90; pulse 82, regular. He was traveling after this to his home in Oklahoma. He was last seen in July, 1938.

COMMENT

On the day of entrance the physical examination revealed a large cardiac dullness and distant heart sounds. Pneumonic signs resembled a bronchopneumonia and pleural effusion on the right. The x-rays also brought this out. The E. K. G. revealed low voltage of all complexes in all leads. Rate 130, regular.

With the pleural effusion, the pericardial effusion and increased venous pressure, a liver edge which was below the costal margin, were we dealing with a case of Pick's disease? It is stated by Paul White, in discussing pericarditis, that over 50 per cent of the cases are missed, because of no symptoms or signs. An increased venous pressure, an increased liver dullness, ascites, with a few or no heart signs in young persons, suggests Pick's disease. (As for effusions, it may be stated here that the normal amount of pericardial fluid is 25 to 50 cubic centimeters.^{3,4,5} The usual abnormal amount is less

* Other slides presented following phases:
Slide 6. Complete drainage and clearing.
Slide 7. Healed wound from pericardial drainage.
Slide 8. Healed wound from left pleural drainage.

than 500 cubic centimeters, being the least that can be recognized (Cabot). Amounts of over 4,000 cubic centimeters have been reported.) However, this patient's signs and symptoms could not be brought under the term "Pick's disease."

To seek further, a paracentesis was done, and purulent fluid was obtained from the pericardial sac. Another was done and a clear fluid came from the right pleural cavity. The former had an organism, present on culture (hemolytic *Staphylococcus aureus*). The fluid from the pleural cavity was sterile. We were certain then of a suppurative pericarditis, caused by the above organism.

SUMMARY

In summarizing the diagnosis considered in this interesting case, we have, first, cellulitis of the right neck; second, an aleukemic phase of a usual myelogenous leukemia with cardiac and pericardial complications; third, Pick's syndrome; and fourth, the correct diagnosis, namely, acute suppurative pericarditis with an initial leukemoid blood picture, which was apparently subsequent to a bronchopneumonia. The offending organism was a hemolytic *Staphylococcus aureus*. It is of unusual interest that, although the pericardial effusion was purulent, both the right and left pleural effusions were clear and sterile originally. The right effusion cleared up on repeated simple aspiration. It was only after operative intervention with pericardial drainage that the left pleural effusion became purulent and required open drainage. Complete recovery followed open surgical drainage of both the pericardium and the left pleural sac; but had the infection been generalized rather than localized to the lungs and pericardium, the outcome would probably not have been so fortunate.

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MINUTEMEN OF AMERICAN MEDICINE*

Q. Why was a question and answer digest prepared? [on the aims and objectives of the National Physicians Committee for the Extension of Medical Service.]*

A. The task that has been undertaken is of vast and vital importance. It affects every practicing physician. It is essential that every doctor understand the purpose and the methods of the institution.

Q. What fact or factors were responsible for the establishment of the National Physicians Committee for the Extension of Medical Service?

A. The twenty-five year trend in political thinking and legislation affecting medicine, medical practice and health.

Q. What was the influence of the report of the Interdepartmental Committee to Coordinate Health and Welfare Activities?

A. It resulted directly in the introduction in the Senate of the Wagner National Health Bill.

*A Question and Answer Digest and Exposition of the Origin, the Aims, Purposes and Methods of the National Physicians Committee for the Extension of Medical Service. (For editorial comment in this issue, see page 105.)

Note.—The *Detroit Medical Bulletin* has named the members of the National Physicians Committee for the Extension of Medical Service "The Minutemen of American Medicine." The designation is self-explanatory.

The institution was officially established as a nonprofit, nonpolitical trust on November 18, 1933.

For action by Council of California Medical Association concerning National Physicians Committee (approval) see in this issue on page 131, item 45.

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